of the store of energy within the muscle and the clogging of the muscular machine by the metabolic products.[1] During exercise the steady outflow of impulses to the muscles, the complex neural processes involved in any voluntary movement and the focussing of the attention upon the exercise must involve considerable expenditure. Hence it is easy to see that fatigue could be induced with very little exercise unless the walking is carefully gauged.

I have attempted to visualize exercise from a different phase than it is usually attacked and to appreciate the significance of the various symptom-complexes which may be produced in consequence of a pathologic process affecting the lungs.

POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNGS.

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RATHER singularly the subject of massive collapse of the lungs as a postoperative complication has scarcely been mentioned in American medical literature up to the present time, and only by English or Canadian authors. The importance of its consideration is accentuated by the fact that it undoubtedly occurs very commonly both in civil and military practice, and also because it is frequently confused with other more or less common postoperative pulmonary complications such as pneumonia, pleuritis, pleural effusions, etc.

Attention was first drawn to the occurrence of massive collapse of the lungs by W. Pasteur,[1] who cited 34 cases as occurring with postdiphtheritic paralysis of the diaphragm or other accessory respiratory muscles in 1890. It is interesting to note that Pearson-Irvine, in 1876, made the observation on cases which undoubtedly were cases of massive collapse, "That the physical changes observed in the lungs were the result of paralysis of the muscles concerned in the elevation and expansion of those parts." In a later series of 64 cases of postdiphtheritic phrenic paralysis, with 15 fatal results and with autopsies on 8 of these, Pasteur was able to demonstrate the gross pathology in 5, the others proving to be cases of bronchopneumonia. The cases were all bilateral, in a more or less advanced collapse, and all presenting the same characteristics *en grosse,* the parts being entirely devoid of air, of a deep, definitely circumscribed blue color and sinking entirely in water. Pasteur noticed the similarity in the symptomatology between numerous clinical cases developing postoperatively and these cases of postdiphtheritic paralysis with collapse of the lungs. In 1908, in the Bradshaw lecture before the Royal College of Physicians, he drew attention to the condition, since which time he has encountered an increasing number of these cases and a corresponding diminution in those of postoperative pneumonia.[2] The clinical features were accurately described, the diagnosis elaborated, and in 1914[2] he published an article drawing attention to its frequency. Of 201 lung complications out of 3559 cases, in the Middlesex Hospital between 1906 and 1910, he found 12 cases of massive collapse, with 1 death; in frequency less than pneumonia, bronchitis or dry pleurisy, but more common than embolism, abscess or pleural effusion. It was found to occur with all methods of anesthesia, and occurred following operations on all areas of the abdomen.

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[1] Internat. Jour. Med. Sc., 1890.

About the same time Pasteur wrote his contribution in 1914, Dingley and Elliott[3] published an article in the *Lancet,* inspired by Sir Rickman Godlee, who showed them several cases encountered in his practice. During a period of two years the writers observed 11 cases, all of which followed abdominal operations. Their contributions to the literature is noteworthy chiefly on account of their consideration of the possible causes of this condition. They recalled Lichtheim's[4] observations, made in 1878, in which he had produced a condition similar, in rabbits by introducing laminaria plugs into the bronchi, resulting in a collapse of the lung tributary to the bronchus, which was the subject of the experiment. The alveoli were passively closed by absorption of the air content into the circulating blood, producing thereby a negative pressure, causing the neighboring organs to be dislocated. Dingley and Eliott conclude that in man, in addition to the comparative immobility of the thoracic wall, secretion blocks the smaller bronchioles, with the result that collapse ensues, just as in Lichtheim's animal experiments. Pasteur postulated an active collapse of the lungs, with an absence of any obstruction to the air passages, which he thought was induced by reflex inhibition of the diaphragm. He believed the collapse, to put it simply, to be due to alveolar expulsion rather than alveolar absorption.

Rose Bradford[5] devotes a chapter to the consideration of this subject in a recent *System of Medicine,* in which he refers largely to previous articles written by him in 1918–1919.[6] His experience was largely with military practice, with a large incidence, particularly in gunshot wounds of the chest, in which injuries he believes it occurs in 5 to 10 per cent of the cases. He points out that in gunshot wounds of the head or arms massive collapse has not been known to occur, but is occasionally seen as a complication in wounds of the buttocks, pelvis and thighs, assuming therefrom that the degree of immobilization is a factor in its production.

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[2] British Jour. Surg., vol. **1.**

[3] Lancet, London, May, 1914.

[4] Arch. f. exper. Path. u. Pharmakol., **10,** 54.

[5] Oxford Loose-leaf Medicine.

[6] Quart. Jour. Med., 1918–1919.

Briscoe,[7] in 1920, emphasized the effect of deficient respiratory excursion and a recumbent posture in causing diaphragmatic fixation with pleuritis, and coincidently pulmonary deflation.

Recently, Scrimger[8] in his article speculates on the various theories heretofore promulgated regarding the causation of massive collapse, and adds his belief that the lesion is probably due to an abdominal interference with the vagus control, causing a contraction of the muscular elements of the lung, aided by the subsequent collection of mucus in the bronchi sufficient to prevent the egress of air and leading to absorption of the alveolar air content and ultimately collapse. His article is interesting, particularly on account of the roentgenographic studies, which in some of his cases show most extravagant alterations in the relationship between the intrathoracic organs.

It is my belief that various factors may produce the lesion and that they are probably never entirely single, except in postdiphtheritic paralyses, in which the immobility of the diaphragm is so extreme as to be strikingly conclusive as to its being the cause. One must bear in mind that on the one hand Lichtheim's experiments are difficult to refute with his careful experimental technic, whereas on the other hand the evidence offered by postdiphtheritic phrenic paralysis is equally incontrovertible. There is no doubt that the recumbent position, as emphasized by Rose Bradford and Briscoe, is an important subsidiary factor as well as deficient aëration of the lungs. It is probable that in civil practice the incidence is more common in abdominal surgery, leading to more or less fixation of the diaphragm, due to a reflex inhibition, with an effort to cause a splinting action on account of the neighboring trauma. The theory that the recumbent posture is a factor is corroborated by the nonoccurrence of massive collapse in injuries not requiring immobilization of the body such as injuries to the head and arms, as observed by Rose Bradford. The admonition to take deep breathing exercises systematically after operations would not only promote aëration of the more distal parts of the bronchial tree, but would also tend to reduce the immobility of the perithoracic musculature. I think it reasonable to assume that with the lack of mobility in the accessory muscles of respiration and the diaphragm, pulmonary expansion and retraction are necessarily limited. Mucus is formed and not expelled, causing an obstacle to the ingress of air into the smaller bronchioles, leading to ultimate alveolar absorption of the air into the circulation.

Bronchial obstruction, therefore, and muscular immobility tend to bring about this condition, one factor or the other predominating, however, in individual cases, leading to alveolar absorption, and finally the condition of massive collapse.

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[7] Quart. Jour. Med., 1920.

[8] Surg., Gynec. and Obst., No. 6, **32**.

The extent and the site of involvement in massive collapse vary considerably. In most cases there is only a partial involvement of one of the lower lobes of the lungs; in others the condition is more extensive, involving a whole lobe or even an entire side. It is rather frequently bilateral. The noteworthy feature is the fact that in unilateral trauma collapse occurs not only on the side affected but oftentimes on the contralateral side, as was pointed out by Rose Bradford and others.

Physical Signs and Symptoms. The physical signs and symptoms of massive collapse are so distinctive that it is curious that the condition has not been recognized more commonly and described more frequently. A careful study of the case will make differentiation from conditions simulating it very easy. The exciting factor varies a great deal, and any trauma, either accidental or otherwise, necessitating more or less immobilization of the body may bring about the condition. It has been known to follow all methods of anesthesia, has occurred with local anesthesia and without any anesthesia at all. Aside from military practice I believe that in civil life it will be found that abdominal operation is the most frequent cause of the condition, on account of its immediate effect in requiring immobilization, producing immobility of the diaphragm and deficient aëration of the lungs. The condition may develop within a few hours or as late as one week after the exciting trauma. The onset is sudden, the course either rapid or at times protracted and resolution either prompt or slow. The degree of temperature is usually very moderate, but may be as high as 103° or 104°F, the coincidence of inflammatory phenomena probably influencing the height of the temperature curve. The respiration rate is increased by the immobility of the affected part, by an accompanying pleuritis or by toxic conditions incident to inflammatory complications. The pulse-rate is found to be more or less in direct relationship with the respiratory and thermic changes, but is less marked in uncomplicated cases, as would be naturally inferred.

On examining the chest one is impressed by the diminished or absent excursion of the chest wall over the affected area. The cardiac impulse is displaced toward the affected side and is as marked in the left-sided cases as in those occurring on the right side, the apex having a tendency to tilt outward and upward, so that the apex-beat may be felt in the axilla. In right-sided affections the impulse may be felt at the tip of the sternum or to the right of it. These signs are corroborated by roentgenographic study, the heart retraction being most marked, the dome of the diaphragm ascending to an unusual degree and the pulmonary area appearing partially or totally collapsed. In bilateral affections the displacement of the heart is absent, but the high position of the diaphragm and the collapse of the lungs are easily manifest. On palpation the intercostal spaces on the affected side are found to be narrowed, leading to a relative approximation of the ribs. The percussion note over the affected area is dull and may approach flatness. Rose Bradford points out that in left-sided cases the lower part of the chest wall is highly resonant, due to the abnormally high level of the diaphragm, the liver interfering with this symptom in right-sided cases. Breath sounds and fremitus are usually increased, sometimes enormously so, but may be diminished or absent. The transmission of voice sounds may be so intense as to approach whispered pectoriloquy.

Bronchophony was present in all of my cases, and in no case was there diminution of the breath sounds or fremitus. No doubt the alteration in the transmission of sounds is due to the relative proximity of the affected area to sound-conducting bronchial tubules. Rales are usually absent in the early stages, but supervene as the case progresses, probably due to a bronchitis, resolution or an occasional pneumonia developing. In a great many cases a pleural friction rub is plainly audible. In the later stages of the condition, when expectoration is rather profuse and resolution occurs, rales are more frequent. As resolution occurs the heart gradually returns to its original position, the lungs slowly expand and the diaphragmatic dome flattens out.

The extent of the symptoms and the ease of recognition depend, of course, upon the amount of lung tissue involved and as to whether the condition is unilateral or bilateral. It is readily understood also that the influence of the condition on the patient's economy is dependent largely upon complicating factors as well as upon the extent of the lesion. Dyspnea may be moderate or extreme, as it usually is in bilateral cases. The cough is slight, as a rule, in the beginning, with a rather scant expectoration, but in the later stages it is accompanied by an expectoration of profuse mucopurulent sputum. My experience bears out the previous observation that the sputum is rarely or never bloody—an important consideration in the differentiation between pneumonia or infarct and collapse.

Bronchitis, pleurisy and pneumonia are recognized as complications. Effusion has been known to follow pleurisy. A differentiation must be made between pneumonia, hypostatic congestion of the lungs, embolus, infarct, pleuritis (with or without effusion), hemothorax and massive collapse. When one bears in mind the outstanding pathognomonic signs of massive collapse, confusion with other conditions is difficult. One must bear in mind that the affected side is retracted and does not expand with inspiration. The diaphragmatic and cardiac displacement is extreme and the general symptoms invariably less severe than with pneumonia or embolus. The very marked dulness, the extreme increase in the breath sounds (as usually noted), the scant expectoration, the comparative absence of constitutional signs in the non-occurrence of complications, accompanied by displacement of the heart and diaphragm and roentgenographic studies, easily establish the diagnosis.

The prognosis is invariably good, but bilateral cases, or cases affecting more than one lobe, are more apt to be fatal, particularly in debilitated subjects.

A brief case report of the cases occurring in our practice is submitted, chiefly because of the interesting autopsy findings in the fatal case:

Case I.—Mrs. D., aged thirty-eight years, was admitted to St. Mary's Hospital, Duluth, November 19, 1920. Her general condition was good.

She was operated upon November 20, by Dr. W. A. Caventry, for the removal of an ovarian cyst and appendix. The gall-bladder and stomach were examined and were found normal. Her temperature before operation was 98.6 °F. and the leukocyte count 9000.

November 22 (two days after the operation) the pulse rose rapidly from 90 to 130 and the temperature to 101 °F. The temperature and pulse varied little from this day until November 25, when there was a gradual reduction, day by day, until normal on November 28. The leukocyte count on November 22 was 21,200 and on November 25 it was 14,600.

Physical Findings. There was limitation of motion on breathing of the left side of the chest. There was marked dulness over the lower lobe of the left lung. Bronchial breathing was present over the area of dulness and no rales were heard. The left border of the heart extended nearly to the axillary line and the apex-beat was felt in the fifth interspace, 5 cm. outside the nipple.

There was no change the following day except that the bronchial breathing was further increased.

November 25, when the patient's condition was better, the left border of the heart had approached its normal position, the bronchial breathing had become vesicular and mucous rales were heard.

Case II.—Mr. W., aged forty-nine years, was admitted to St. Mary's Hospital, Duluth, June 23, 1921, with an acute appendicitis. The temperature was 100 °F. and the leukocyte count was 20,000.

He was operated on the same day, by Dr. T. L. Chapman. A gangrenous appendix was removed and was closed without drainage.

June 24, the day following the operation, his temperature rose to 102 °F. and the pulse to 110. The patient was apparently distressed. Examination of the chest showed an area of dulness, approaching flatness, corresponding to the lower right lobe. Moderate bronchial breathing with increased transmission of whispered sounds. The left border of the heart was displaced to the right about 6 cm.

The following day the patient's condition was about the same, but the temperature had risen to 104°F. and the pulse to 120. The physical signs were the same. There was still an absence of any rales. In the evening of June 25 the temperature dropped to 99°F. and the pulse to 85; the patient was apparently much better.

The following morning the right lower chest was resonant, a few rales were heard and the heart approximately was in the normal position.

CASE III.—Mr. E. J. M., aged forty-eight years, was admitted to St. Mary's Hospital, Duluth, April 2, 1921, and was operated on April 6 for a chronic duodenal ulcer. An appendectomy and posterior gastroenterostomy were done.

April 8 the patient suddenly developed acute respiratory distress, with moderate cyanosis. The temperature at that time was 99°F. and the pulse was 110.

April 9 his temperature was 101°F.; pulse, 140; respiration, 38. Physical examination showed the patient to be in severe distress, with difficulty in breathing and in an apprehensive condition. The bases of both lungs expanded poorly. The vocal and tactile fremitus were increased on both sides. Dulness was extreme. On auscultation there was marked increase in the transmitted breath and whispered voice sounds. There was no displacement of the apex-beat. The patient's condition was too grave to permit of roentgenographic studies, became rapidly worse and he died on April 10.

A partial autopsy was permitted, consent being given to open the operative wound. This was found to show moderate healing and was void of any visible evidence of infection. The site of the gastroenterostomy and the appendicular stump were clean and without adhesions. It was necessary to deliver the lungs through the diaphragm, which was done without very much difficulty. The appearance of the lower lobes on both sides was most striking. Definitely circumscribed areas involved nearly the whole of both lower lobes, sharply demarcated from the normal tissues by a deep violet color. The collapsed areas were definitely depressed in relation to normal lung tissue, and contained no air, blood nor pus in the alveolar areas. The upper lobes were normal in appearance and showed no evidence of hypostatic congestion.

Cut sections from the affected area did not float in water.

Microscopical section showed an approximation of the alveolar surface to a marked degree, with a few red and white blood cells in the alveolar spaces. The interalveolar tissue was normal.

The autopsy findings were otherwise entirely negative.

Attention is called to the relative frequency of this condition and the importance of its recognition from a diagnostic standpoint.